Nonsustained Ventricular Tachycardia in Heart Failure

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Summary:

Background: *Heart failure is a common clinical syndrome with a high morbidity and mortality, despite advances in medical treatment. Death from dangerous ventricular arrhythmias is frequently implicated.*

Patients, materials & methods: *Eighty patients with heart failure (HF) (fitting the criteria of heart failure) who were admitted to the medical city teaching hospital during a period of 8 months, were studied for incidence of nonsustained ventricular tachycardia (N.S.V.T.) (detected by Holter monitoring) and its association with the severity of left ventricular dysfunction (measured by ejection fraction), ventricular size (measured by left ventricular end diastolic dimension), and other factors .*

Results: *It was found that out of 80 patients with H.F, 20 patients (25%) have N.S.V.T. N.S.V.T was found to be significantly associated with the severity of H.F. and left ventricular dimension. The arrhythmia was found to be strongly related with hypokalemia. The incidence of N.S.V.T. is less in patients receiving beta-blockers in their treatment regimen.*

Conclusion: *Nonsustained ventricular tachycardia is a common finding in heart failure and is related to the severity of heart failure and other factors related to the disease and its treatment.*

Introduction:

Heart failure is a common clinical syndrome with a high morbidity and mortality despite advances in medical treatment. Death from dangerous ventricular arrhythmias is frequently implicated.

Systolic function of the heart is governed by four major determinants: the contractile state of the myocardium, the preload of the ventricle (the end diastolic volume and the resultant fiber length of the ventricle prior to onset of contraction), the after-load (the impedance to left ventricular ejection), and the heart rate (') Cardiac function may be inadequate as a result of alterations in any one of these determinants. In most instances, the primary derangement is depression of myocardial contractility caused either by loss of functional muscle (due to myocardial infarction) or by processes diffusely affecting the myocardium (Z). The heart may fail as a pump because preload is excessively elevated, such as in valvular regurgitation, or when after load is excessive, such as in aortic stenosis or in severe hypertension. Pump function may also be inadequate when the heart rate is too slow or too rapid (3). While the normal heart can tolerate wide variations in preload, afterload, and heart rate, the diseased heart often has limited reserve for such alterations (4). Cardiac pump function may be supranormal but nonetheless inadequate when metabolic demands or requirements for blood flow are excessive. This situation is termed high-output heart failure and, though uncommon, tends to be specifically treatable. Causes of high-output heart failure include: thyrotoxicosis, beriberi, severe anemia, arteriovenous shunting and Paget's disease of bone (5). When the heart fails, a number of adaptations occur, both in the heart and systemically. If the stroke volume of either ventricle is reduced by depressed contractility or excessive afterload, end diastolic volume and pressure in that

chamber will rise. This increases end diastolic myocardial fiber length, resulting in a greater systolic shortening (Starling's law of the heart). If the condition is chronic, ventricular dilatation will occur 181. Reduced cardiac output, particularly if associated with reduced arterial pressure or perfusion of the kidneys, will also activate several neural and humoral systems like the renin angiotensin aldosterone system and the sympathetic nervous system leading to adaptive changes aiming at improving cardiac output (7, 8). Though these adaptations are designed to increase cardiac output, they may themselves be deleterious. (9)

In patients with congestive heart failure, atrial and ventricular arrhythmias are very common and may be related to electrolytes changes (e.g. hypokalaemia, hypomagnesaemia), the underlying structural heart disease and the proarrhythmic

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effects of increased catecholamines and some drugs (e.g. digoxin) (10, 11). Sudden death occurs in up to 50% of patients with severe heart failure and is often due to ventricular arrhythmias. Frequent ventricular ectopic beats and runs of nonsustained ventricular tachycardia are common findings in patients with heart failure and are associated with an adverse prognosis. (12,13)

Aims of the Study

This study attempts to determine the incidence of nonsustained ventricular tachycardia in patients with heart failure and to determine the factors affecting this incidence.

Patients, Materials and Methods

This study was done from March 2004 to November 2004 in the Medical CityBaghdad teaching hospital. Eighty patients with H.F were included. Their ages ranged between (14 - 85) years with a mean of (54) year. Forty-five patients were males and 35 were females, the male: female ratio was 1.3:1. The duration of symptoms ranged between 3 weeks and 27 weeks with a mean duration of 12 weeks. The diagnosis was made according to the Framingham's criteria of H.F (14) (table 1). Nonsustained ventricular tachycardias (N.S.V.T) were defined as runs of 3 or more ventricular beats lasting less than 30 seconds occurring at rates > 100 beats / min.

Patients with extensive myocardial infarction complicated by acute heart failure within one month were not included in this study, as any arrhythmia which might be recorded could be attributed to ischemia. The patients were evaluated by history and clinical examination, and relevant tests were done that included electrocardiography, chest X-ray, echocardiogrophy, renal function tests and serum sodium (Na^+) and potassium(K^+). Holter monitoring for 24 hrs was done for all patients included in this study by using cardiosoft Hotter seer Ms software version 2b, 2007185-010 revision A.

Data from all patients were tabulated and statistical analysis was done by using student t test, and the results were regarded as statistically significant when the p. value was < 0.05.

Results

Non-sustained ventricular tachycardia was recorded in 20 patients (25%). We studied the relationship between N.S.V.T and several parameters (table 2). We found that the incidence of N.S.V.T was significantly increased with increasing severity of heart failure (as assessed by left ventricular ejection fraction [LVEF]), increasing left ventricular size (as assessed by LV end diastolic dimension [LVEDD]), and low serum potassium levels. The incidence was significantly decreased by the use of Beta blockers. This study found no significant relation between the incidence of N.S.V.T and the etiology of heart failure, serum sodium levels, and treatment with digoxin, angiotensin converting enzyme inhibitors, and spironolactone.

Table (1): Framingham Criteria for Diagnosis of Heart Failure*

Major criteria
- Paroxysmal nocturnal dyspnea or orthopnea
- Neck vein distension
- Rales
- Radiographic cardiomegaly
- Acute pulmonary edema
- S3 gallop
- Increased central venous pressure > 16 cm H_2O
- Circulation time ≥ 25 sec
- Hepatojugular reflux
- Pulmonary odema, visceral congestion, or cardiomegaly at autopsy
- Weight loss ≥ 4.5 Kg in 5 days in response to treatment of heart failure
Minor criteria
- Bilateral ankle edema
- Night cough
- Dypnea on ordinary exertion
- Hepatomegaly
- Pleural effusion
- Decrease in vital capacity by one third from maximal value recorded
- Tachycardia (rate ≥ 120 / min)

* To establish a definite diagnosis of heart failure two major or one major and two minor criteria have to be present concurrently. Minor criteria were acceptable only if they could not be attributed to another medical condition.

Table (2): The Incidence of N.S.V.T in Relation to the Severity of H.F (LVEF), Left Ventricular Size (LVEDD), Serum Potassium and Sodium Levels and Certain Drugs Used in Patients with Heart Failure.

Parameter		Total No. Of patients	No. Of patients With N.S.V.T.	%Of patients With N.S.V.T.	P. Value*
LVEF(%)	20-29	10	7	70%	0.005
	30-39	29	9	31%	
	40-49	41	4	10%	
LVEDD (mm)	58-68	43	3	7%	0.001
	68-77	27	9	33%	
	>78	10	8	80%	
Serum K^+ (Meq/l)	<3.5	19	9	47%	0.01
	3.5-4.5	47	9	19%	
	>4.5	14	2	14%	
Serum Na^+ (Meq/l)	<125	4	2	50%	0.08
	125-135	34	10	29%	
	136-145	36	7	20%	
	>145	6	1	17%	
Beta blocker	Present	16	1	6	0.002
	Absent	64	19	29.68	
Digoxin	Present	28	8	28	0.07
	Absent	52	23	23.07	
ACE inhibitor	Present	75	18	24	0.06
	Absent	5	2	40	
Spironolactone	Present	30	6	20	0.07
	Absent	50	14	28	

* P value < 0.05 was considered statistically significant.

Discussion

The incidence of N.S.V.T in our study was 25% which is less frequent than other study done by (Pocker M et al) [6] that had found that the incidence was 70% in moderate to severe HF. There are many obvious differences in data collection and selection of patients. Advanced cases of HF with minimal duration of one month were pooled in that study in addition to long periods of follow up with abundant use of Holter monitoring, while our study lacks the follow up and we have used 24 hour Holter monitoring once in each patient. Also, we excluded the acute cases of HF which developed within the first month after acute myocardial infarction.

In our study, the risk of developing N.S.V.T was significantly related to increasing severity of heart failure as measured by LVEF (P value 0.005). This result is similar to the results of other studies. For example, Biggar J.t et al showed that N.S.V.T occurred more frequently in patients with LVEF <30% than in patients with LVEF >40% [13]. Also, the study of Marechlinski et al found that low LVEF was an independent risk factor for developing N.S.V.T and for sudden death due to ventricular arrhythmias (').

In our study, the incidence of N.S.V.T was significantly associated with increasing left ventricular size as measured by left ventricular end diastolic dimension (LVEDD) (p value 0.001). This is in accordance with the results of studies done by Massie BM and MeMurray JJ et al who showed that increasing LVEDD

increased the risk of developing N.S.V.T regardless of EF nor the etiology of heart failure (2, 15). Our study showed no significant association of N.S.V.T with the underlying causes of H.F, a result which stands with those obtained from other studies (9, 15). This
observation supports the role of myocardial stretch in the development of arrhythmia, and that mechanical dilation of the human ventricle is known to induce arrhythmia; the underlying ionic mechanisms, however, remain to be clarified. Our data demonstrated that N.S.V.T is significantly associated with hypokalaemia (p value 0.01). It has long been known that even mild hypokalaemia (3-3.5 meq/I) predisposes to cardiac dysrhythmia in patients with H.F (5). Pocker M et al regarded a serum K+ 3.5meq/I is not normal for patients with H.F and that these patients should have their potassium concentration maintained at a level of 4 meq/I or greater especially in patients receiving digoxin (6). This can be attributed to the fact that a decrease in serum K+ causes an increase in the resting potential of cardiac cells and prolongation of both action potentials and refractory periods. Hyponatremia is a marker of renin-angiotensin system activation in patients with H.F, and a low serum Na' is a bad prognostic parameter in H.F (5). Studies done by pocker M et al and Clenand JG demonstrated that a disturbance in this electrolyte is an independent risk factor for developing arrhythmia and should be monitored regularly in patients with H.F (6, 's). In our study, the relation was not statistically significant (p value 0.09). This difference in our study could be attributed to the high number of patients who had low normal or just mildly low serum Na+ levels and only 4 patients had serum Na+ <130 meq/I.

Regarding the type of treatment and its association with the incidence of N.S.V.T our study showed that 15 patient (94%) out of 16 patients who were receiving beta blockers in their treatment regimens had no such arrhythmias and the difference reached statistical significance (p value 0.002). This is in accordance with the results of a study done by Abraham Wt (11) who showed that the use of beta blockers in patients with H.F (NYHA class II and III) resulted in consistent substantial rises in
ejection fraction (EF)(averaging 10% absolute increase) and reduction in left ventricular size and mass, as well as strong reduction in N.S.V.T. This agrees with the hypothesis saying that the chronic elevation of catecholamines and sympathetic nervous system activity cause progressive myocardial damage, leading to worsening left ventricular function and dilation which finally predispose to ventricular arrhythmias.

On the other hand, our study didn't show significant difference with other types of drugs used in the treatment of HF. Other studies, however, showed that the risk of developing N.S.V.T was high in patients who were taking digoxin even in the absence digitais toxicity, and this risk is more in hypokalaemic patients (6. 17, 18). In addition, Rather M et al showed that the use of ACE inhibitors in patients with heart failure reduced the incidence of sudden death due to malignant ventricular arrhythmias and both the frequency and the duration of N.S.V.T were reduced by
about 20% (19).

Conclusions & Recommendations:

1-N.S.V.T is not uncommon in patients with C.H.F.

2-Electrolyte disturbances are common etiological factors for N.S.V.T especially hypokalemia, either due to disease pathophysiological changes or diuretic therapy. It is advisable to keep serum K+ of such patients at levels > 4 meq/I. 3-N.S.V.T is more common in patients with very low EF and very high LVEDD, so the drugs known to prevent the development of remodeling (like ACE inhibitors) should be used early in the disease and in adequate doses.

4-Beta-blockers seem to be protective against development of N.S.V.T and should be used when possible.

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